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Term: Urokinase-type adj plasminogen and chimeric and liver

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USPT	Urokinase-type adj plasminogen and chimeric and liver	28	<u>L5</u>
USPT	Urokinase-type adj plasminogen and chimeric	45	<u>L4</u>
USPT	Urokinase-type adj plasminogen adj activator and transgenic and liver	15	<u>L3</u>
USPT	Urokinase-type adj plasminogen adj activator and transgenic	21	<u>L2</u>
USPT	Urokinase-type adj plasminogen adj activator	165	<u>L1</u>

09186892

L9 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2000 ACS

ACCESSION NUMBER: 2000:210343 CAPLUS

DOCUMENT NUMBER: 132:247157

TITLE: Urokinase-type plasminogen
activator/RAG-2mouse with mammalian repopulating hepatocytes
for infection by hepadnaviruses

INVENTOR(S): Rogler, Charles E.

PATENT ASSIGNEE(S): Albert Einstein College of Medicine of Yeshiva
University, USA

SOURCE: PCT Int. Appl., 41 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000017338	A1	20000330	WO 1999-US21838	19990916
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.:

US 1998-156892 19980918

US 1999-344189 19990624

AB This invention provides a chimeric **mouse** liver model system for mammalian hepatitis. A method is provided for repopulating degenerated liver of immunotolerant mice which lack mature B and T lymphocytes with xenogenic mammalian hepatocytes, particularly primate hepatocytes to generate chimeric mice. In addn., a method of generating a human hepatitis virus-infected chimeric **mouse** is provided. A preferred xenogenic primate hepatocyte is derived from human, chimpanzee, or baboon. These chimeric mice are useful in the investigation of host and viral mechanisms detg. hepadnaviral persistence and hepatocarcinogenesis. Methods for monitoring the development of hepatitis and hepatocellular carcinoma as well as methods for testing and screening anti-viral and anti-cancer compds. with this model system are also provided.

REFERENCE COUNT: 3

REFERENCE(S):

- (1) Gupta, S; AM J PHYSIOL PT 1 1999, V277(6), PG1097 CAPLUS
- (2) Petersen, J; PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF USA 1998, V95(1), P310 CAPLUS
- (3) Ponzetto, A; ITAL J GASTROENTEROL 1991, V23(8), P491 MEDLINE

QH.N26

L8 ANSWER 10 OF 15 MEDLINE

DUPLICATE 5

ACCESSION NUMBER: 95281572 MEDLINE

DOCUMENT NUMBER: 95281572

TITLE: Complete reconstitution of **mouse liver**
with xenogeneic hepatocytes.

AUTHOR: Rhim J A; Sandgren E P; Palmiter R D; Brinster R L

CORPORATE SOURCE: Department of Animal Biology, School of Veterinary
Medicine, University of Pennsylvania, Philadelphia 19104,
USA.

CONTRACT NUMBER: HD-23657 (NICHD)

CA-38635 (NCI)

HD-09172 (NICHD)

SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE
UNITED STATES OF AMERICA, (1995 May 23) 92 (11) 4942-6.

Journal code: PV3. ISSN: 0027-8424.

PUB. COUNTRY: United States

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals; Cancer Journals

ENTRY MONTH: 199509

AB We have developed a system for studying hepatocellular growth potential
in

which liver cells are introduced into the diseased livers of
albumin-urokinase (Alb-uPA) **transgenic** mice. To use
this system to study xenogeneic cell transplantation, rat **liver**
cells were introduced into immunotolerant Alb-uPA
transgenic mice. In regenerated recipient livers, up to 100% of
hepatocellular gene expression was of rat origin, demonstrating the
creation of a functional **mouse liver** in which
parenchyma is derived from xenogeneic (rat) hepatocytes. Immunotolerant
Alb-uPA **transgenic** mice provide a tool for studying
hepatocellular biology of any species, including humans, in a controlled
experimental setting.

QU. N26

L8 ANSWER 14 OF 15 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 6
ACCESSION NUMBER: 1991:431173 BIOSIS
DOCUMENT NUMBER: BA92:87338
TITLE: COMPLETE HEPATIC REGENERATION AFTER SOMATIC DELETION OF AN
ALBUMIN PLASMINOGEN ACTIVATOR TRANSGENE.
AUTHOR(S): SANDGREN E P; PALMITER R D; HECKEL J L; DAUGHERTY C C;
BRINSTER R L; DEGEN J L
CORPORATE SOURCE: LAB. REPRODUCTIVE PHYSIOL., SCH. VET. MED., UNIV.
PENNSYLVANIA, PA. 19104.
SOURCE: CELL, (1991) 66 (2), 245-256. Q4573.C38
CODEN: CELLB5. ISSN: 0092-8674.
FILE SEGMENT: BA; OLD
LANGUAGE: English


AB We previously demonstrated that expression of an albumin-urokinase
-type plasminogen activator (Alb-uPA
) fusion construct in transgenic mice resulted in elevated
plasma uPA concentration, hypofibrinogenemia, and neonatal
hemorrhaging. Two lines of Alb-uPA mice were established in
which only one half of the transgenic pups died at birth;
surprisingly, plasma uPA concentrations in survivors gradually
returned to normal by 2 months of age. The basis for this phenomenon is
DNA rearrangement within hepatocytes that affects the transgene tandem
array and abolishes transgene expression. Transgene-deficient cells
selectively proliferate relative to surrounding liver, and this
process culminates in replacement of the entire liver by clonal
hepatic nodules derived from transgene-deficient progenitor cells. In
some cases as few as two nodules can reconstitute over 90% of liver
mass, highlighting the remarkable regenerative capacity of individual
liver cells.

L6 ANSWER 8 OF 63 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 3
AN 1999:220856 BIOSIS
DN PREV199900220856
TI Zonal regulation of gene expression during liver regeneration of
urokinase
transgenic mice.
AU Locaputo, Stephanie; Carrick, Terri L.; Bezerra, Jorge A. (1)
CS (1) Division of Gastroenterology and Nutrition, Children's Hospital
Medical Center, 3333 Burnet Ave., Cincinnati, OH, 45229 USA
SO Hepatology, (April, 1999) Vol. 29, No. 4, pp. 1106-1113.
ISSN: 0270-9139.
DT Article
LA English
SL English

DUPLICATE 7

L6 ANSWER 16 OF 63 MEDLINE
AN 1998442697 MEDLINE
DN 98442697
TI Selective repopulation of normal mouse liver by
Fas/CD95-resistant hepatocytes.
AU Mignon A; Guidotti J E; Mitchell C; Fabre M; Wernet A; De La Coste A;
Soubrane O; Gilgenkrantz H; Kahn A
CS INSERM U 129 ICGM, Universite Paris V Rene Descartes, Paris, France.
SO NATURE MEDICINE, (1998 Oct) 4 (10) 1185-8.
Journal code: CG5. ISSN: 1078-8956.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199901
EW 199901

L6 ANSWER 12 OF 63 BIOSIS COPYRIGHT 2000 BIOSIS
AN 2000:87110 BIOSIS
DN PREV200000087110
TI **Mouse** liver tumorigenesis: Models, mechanisms, and relevance to
human disease.
AU Fausto, Nelson (1)
CS (1) Department Pathology, University Washington School Medicine, C-516
Health Sciences Building, Seattle, WA, 98195-7470 USA
SO Seminars in Liver Disease, (1999) Vol. 19, No. 3, pp. 243-252.
ISSN: 0272-8087.
DT General Review
LA English

L9 ANSWER 1 OF 4 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 2001:397008 BIOSIS
DOCUMENT NUMBER: PREV200100397008
TITLE: **Hepatitis C** virus replication in mice with
chimeric human livers.
AUTHOR(S): Mercer, David F.; Schiller, Daniel E.; Elliott, John F.;
 Douglas, Donna N.; Hao, Chunhai; Rinfret, Aline; Addison,
William R.; Fischer, Karl P.; Churchill, Thomas A.; Lakey,
Jonathan R. T.; Tyrrell, David L. J.; Kneteman, Norman M.
(1)

CORPORATE SOURCE: (1) Department of Surgery, Surgical-Medical Research
Institute, University of Alberta, Edmonton, AB:
nkneteman@cha.ab.ca Canada

SOURCE: Nature Medicine, (August, 2001) Vol. 7, No. 8, pp.
927-933.

print.
ISSN: 1078-8956.

DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Lack of a small animal model of the human **hepatitis C** virus
(HCV) has impeded development of antiviral therapies against this
epidemic

infection. By transplanting normal human hepatocytes into SCID mice
carrying a plasminogen activator transgene (Alb-uPA), we generated mice
with chimeric human livers. Homozygosity of Alb-uPA was associated with
significantly higher levels of human **hepatocyte** engraftment, and
these mice developed prolonged HCV infections with high viral titers
after
inoculation with infected human serum. Initial increases in total viral
load were up to 1950-fold, with replication confirmed by detection of
negative-strand viral RNA in transplanted livers. HCV viral proteins were
localized to human **hepatocyte** nodules, and infection was
serially passaged through three generations of mice confirming both
synthesis and release of infectious viral particles. These chimeric mice
represent the first murine model suitable for studying the human
hepatitis C virus